Modern Concepts of Cardiovascular Disease

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THE HEART IN ACUTE INFECTIONS

Circulatory disturbances arising during or following bouts of acute infections may be related to injury of the heart but frequently depend upon failure of the peripheral vascular mechanism. The effects of fever cannot be readily distinguished from damage caused by toxins elaborated by the organism concerned. Cellular injury varying from cloudy swelling to more serious types of degeneration and cellular infiltrations, chiefly around blood vessels, disturb the functions of the vascular bed and the myocardium. The intrinsic neuromuscular apparatus of the heart is particularly susceptible to injury in certain infectious diseases where toxic substances enter the blood stream, e. g., diphtheria. Fortunately, most of the functional disturbances are temporary and subside when the period of active infection has passed.

The usual symptoms and signs of chronic congestive heart failure are not to be expected during the course of an acute infection. In severe pneumonia and some other infections an acute type of myocardial failure is not uncommon, and in typhoid fever or other protracted fevers a high venous pressure with anasarca may supervene. In rheumatic fever, pancarditis may be accompanied by marked signs of congestive failure.

During acute infections the patient seldom complains of symptoms referable to the circulatory system. Precordial pain or vomiting may suggest acute pericarditis. Frequent observations at the bedside, however, will help the physician to detect the evidences of circulatory failure. A feeble, rapid pulse, falling blood pressure and diminished sounds at the apex may indicate relaxed peripheral vessels or failing myocardium. Dilatation of the entire heart or of certain chambers may occur. Disturbances in pace, rhythm or heart block point to injury of the heart. These signs with cyanosis and pallor are among those most commonly observed. Cyanosis may be limited to the nails and mucous membranes with a background of pallor. The cyanosis in pneumonia and other pulmonary infections does not necessarily indicate cardiac failure. Tachycardia disproportional to the height of the fever, according to the pattern common to many infectious diseases, should direct attention at once to the circulatory system. Bradycardia occurring suddenly in any of the infectious diseases is presumptive evidence of heart block. A dicrotic pulse is common to many infections with a relatively slow pulse and low pulse pressure, and is not, of itself, a sign of great significance. A failure of the cardiac sounds may be the first sign of serious complications. In severe cases, the sounds may be faint or absent. In pneumonia a diminishing pulmonic second sound, a fall in the peripheral blood pressure, a rise in venous pressure and increased cyanosis point to a fatal outcome.

Some of the murmurs observed during the course of an acute infection may reasonably be associated with valvular disease, but more often they are related to failure of "streamlining" resulting from disproportion between the size of the valves and the chambers on either side or to lack of tone in the myocardium. In rheumatic fever and other acute infections the systolic murmur at the apex is probably caused by slight but sufficient dilatation of the left ventricle to weaken the seat of the mitral valve and permit regurgitation of a part of the charge of blood into the left auricle during ventricular systole. It is of interest that this systolic murmur often disappears after the infection subsides.

The nature of the infectious disease should keep the physician on the alert for special cardiac complications. The great frequency of localizations in the heart in rheumatic fever, and the important bearing these involvements have on the after-life of the individual, require observation until long after the acute symptoms have subsided. When pancarditis is present, the symptoms and signs are usually obvious. In the milder forms, the signs are more elusive. The sounds may be lacking in tone, and systolic murmurs may be observed. A persistent tachycardia may be the only sign. Bradycardia or irregularities in rhythm suggest damage to the intrinsic neuromuscular mechanism. Heart block or the more subtle changes in the myocardium and conduction system may only be determined after an electrocardiogram has been made.

In diphtheria, the onset of signs of cardiac damage may be swift and the result disastrous. Various observers state that cardiac complications arise in from ten to twenty per cent of cases, although electrocardiographic studies may show minor evidences of injury in a higher percentage. These complica-

tions appear at any time from the end of the first to the fifth or sixth week, but more frequently during the second and third week after onset. Such signs as feebleness of sounds and impulse over the heart, cyanosis and pallor and enlargement, are those most frequently observed. Irregularities in rhythm, particularly dropped beats, recognized by absence of sounds during the pause at the heart, and sudden changes in pace where the minute-rate is halved, strongly suggest the more marked degrees of auriculoventricular or sinoauricular block. A gallop rhythm may be the sign of damage in the conduction system or ventricular muscle, which could be verified by the discovery of an alternating pulse or by changes in the ventricular complexes in the electrocardiogram.

In scarlet fever, as in diphtheria, the earlier cardiac signs are probably those associated with the effects of a soluble toxin. Later, when the febrile stage has passed, the perivascular infiltration and exudation may depend upon the hypersensitive state, as has been proposed by Swift and others. Endocarditis and pericarditis may be observed. While the conduction system is attacked less frequently than in diphtheria, it is not uncommon to observe sinoauricular or auriculoventricular block of varying degree in severe cases. The electrocardiogram may show altered Q R S deflections, perhaps, with low voltage and with abnormalities in the T wave.

Influenza may show its effects on the heart early or late. The relatively slow pulse may persist during the stage of pulmonary inflammation. Patients sometimes die with extensive pulmonary edema and cyanosis with a pulse rate of 100 to 110 per minute. The sounds are frequently diminished in fatal cases. Cyanosis is occasionally extreme and during the pandemic, heliotrope hues were noted. After the active stage has passed, the vagal tone may be very marked for a long period, with bradycardia, sinus arhythmia and extrasystoles.

Tuberculosis affecting the pericardium resembles other acute infections in this structure, with or without friction sounds, distant and rapid cardiac sounds, feeble pulse, frequently accompanied by murmurs and enlarged area of dulness. The diagnosis should be suspected in patients with other evidences of tuberculosis or in those in whom no clear antecedent history of rheumatic fever, scarlet fever or other infections prone to affect the pericardium, has been established. Tuberculosis seldom affects the myocardium except by direct extension, and very rarely the valves.

The valves may rarely be attacked during the course of other general infections caused, notably, by such organisms as the pneumococcus, staphylococcus, gonococcus, meningococcus, typhoid bacillus and Pfeiffer's bacillus. Under such circumstances the symptoms and signs are those of an acute bacterial endocarditis with septicemia or septicopyemia manifested by fever, usually leucocytosis, purpura and embolism, localized either in the pulmonary or arterial circuit depending on the side of the heart involved.

The cardiac complications in diphtheria account for most of its fatalities. Early adequate treatment of diphtheria will lessen the chance of cardiac damage. When convalescence is well established, the danger from heart block is very slight but occasionally sudden death occurs from this disturbance or perhaps from ventricular fibrillation. Patients have been known to show persistent heart block for many years after an attack. In other infectious diseases such as pneumonia, typhoid fever, influenza, scarlet fever and the like, the heart and general circulation rarely show signs of serious damage after the infection subsides. The patient with pneumonia may die at the height of the infection with signs chiefly of right cardiac failure. The valvular localization in scarlet fever may be discovered months after the acute stage, leading subsequently to impaired physical activity and a shortened life. Rheumatic fever, with its repeated insults, is notorious for the insidious train of late effects in the heart which makes cardiac cripples of many during the productive period of life, with a consequent shortening of life. The prognosis in acute and subacute bacterial endocarditis with any type of organism is almost hopeless.

The treatment of the cardiac manifestations of acute infections is chiefly supportive. In diphtheria and in other diseases where specific antitoxic sera are available, early and adequate treatment offers the greatest security. Where the temperature is high or perspiration is excessive, increased intake of fluids and chlorides is important. Nourishment should be adequate and easily digestible, especially in the more protracted fevers. Extra strain on the heart should be prevented by control of distention and by removal of excessive fluid in the pleural cavity or pericardial sac. Concentrated glucose solutions given intravenously at short intervals may be of decided benefit to the heart in diphtheria, pneumonia and other infections. Care should be taken not to load the circulation with large amounts of fluid intravenously. Bleeding may be of value occasionally in pneumonia when the right heart begins to fail. The tendency to employ digitalis or strophanthin in all patients with any type of circulatory failure is to be deplored. There is little evidence that digitalis is indicated as a routine in pneumonia, but may be of some value in patients known to have cardiac disease before the onset. Excessive use of this drug may "poison" the heart at the time of the greatest need, with untoward results. If any drug of this group is to be of benefit it should probably be used when the pulmonic second begins to diminish and the blood pressure begins to fail; then digitalis or strophanthin should be given intravenously and repeated as indicated. If strophanthin is used, the single dose should not be greater than 0.00025 gm. (1/250 grain). Caffein is a circulatory stimulant of great value and may help a patient past a critical stage. Again, the dosage must be adequate and often repeated. Lastly, the physician should see that rest and sleep are secured, which may do more for the circulation than any particular method of treatment.

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